## Oculomotor capture and inhibition of return: Evidence for an oculomotor suppression account of IOR

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ORIGINAL ARTICLE

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# Oculomotor capture and Inhibition of Return: Evidence for an oculomotor suppression account of IOR

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**Abstract** Previous research has shown that when subjects search for a particular target object the sudden appearance of a new object captures the eyes on a large proportion of trials. The present study examined whether the onset affects the oculomotor system even when the eyes move directly towards the target. Using a modified version of the oculomotor paradigm (see Theeuwes, Kramer, Hahn, & Irwin, 1998) we show that when the eyes moved to the target object, subsequent saccades were inhibited from moving to a location at which a new object had previously appeared (inhibition-of-return; IOR). Whether or not a saccade to the onset was executed had no effect on the size of the inhibition. In particular conditions, the trajectories of saccades to the target objects were slightly curved in the opposite direction of the onset. The data are interpreted in the context of a novel hypothesis regarding oculomotor IOR.

## Oculomotor capture and Inhibition of Return

When observers visually search for a particular target object, they typically make rapid eye movements (saccades) in order to examine different parts of the visual scene. A number of studies have shown that the appearance of a new object (onset) in the visual scene affects the oculomotor system even when the onset is irrelevant for the task at hand. First of all, the onset often elicits the execution of a saccade in its direction. For example, Theeuwes, Kramer, Hahn, Irwin and Zelinsky (1999; also see Theeuwes, Kramer, Hahn & Irwin, 1998; Irwin, Colcombe, Kramer & Hahn, 2000) presented participants with displays containing six gray circles spaced equally around an imaginary circle. After one second all of the circles except one changed into red. On half of the trials an additional irrelevant red circle (an abrupt onset) was added to the display simultaneously with the color change of the distractors. Participants were required to move their eyes to the uniquely colored gray circle. The results showed that, even though the onset was never relevant to the task, the eyes initially went toward the onset in about one-third of the trials.

A number of other studies have shown that the oculomotor system may be affected by an onset even on those trials in which the eyes move directly to the target. Using the oculomotor paradigm of Theeuwes et al. (1998; 1999) Godijn and Theeuwes (2002) and Irwin, Colcombe, Kramer and Hahn (2000; Exp. 1) showed that latencies of saccades that went directly to a uniquely colored target were about 20 ms higher when an onset was presented than when it was not. One possible explanation for this saccade latency difference is that it took longer to locate the target when an onset was present, due to filtering costs associated with the requirement to ignore the onset. This filtering costs hypothesis has also been suggested by Folk and colleagues (e.g., Folk & Remington, 1998) to explain the finding that manual reaction times (RTs) to color singleton targets are higher when an onset is presented than when it is not (e.g., Theeuwes, 1991, 1992). Although the filtering costs hypothesis was originally intended to explain slowing of covert (i.e., without eye movements) orienting, it might also be used to explain slowing of overt orienting (eye movements). These filtering costs are assumed to be non-spatial, that is, there is no actual shift of orienting to the location of the onset. Instead, the onset first needs to be 'filtered out' before a shift of orienting toward the target can occur. Since the filtering costs hypothesis assumes a non-spatial cost associated with the presentation of the onset it cannot explain why onsets often capture the eyes. A more plausible explanation for these effects of onsets on the oculomotor

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system is that the presentation of the onset initiates the programming of a saccade to its location and that this saccade program competes with the programming of a saccade to the target. According to a number of models (e.g. Trappenberg et al., 2001; Kopecz, 1995; Godijn & Theeuwes, 2002) saccade programming occurs in a common saccade map in which activation at a specific location in the map spreads to neighboring locations, but inhibits activation at distant locations. These models assume that endogenous activation (target-related activation) and exogenous activation (onset-related activation) are integrated in the saccade map. According to Godijn and Theeuwes (2002) when the onset-related activation is strong enough to reach an activation threshold the saccade is directed toward the onset; if the onset-related activation is not strong enough to reach threshold the eyes move toward the target, but with a higher latency due to lateral inhibition from the onset-related activation.

The present study examines whether onsets activate an oculomotor program even when the eyes are not captured by the onset, but instead move directly to the target. To examine this issue we use the inhibition-of-return effect (IOR; Posner & Cohen, 1984). In the typical IOR study (see Klein, 2000 for a review) subjects are required to fixate a central fixation point and to ignore the onset of an uninformative peripheral cue. After a variable stimulus-onset-asynchrony (SOA) relative to the peripheral cue a target is presented at the same location as the onset or at a different location. Subjects are typically required to respond as quickly as possible by pressing a key (or executing a saccade to the target). If the SOA is large enough (at least 200 to 300 ms) responses are slower when the target is presented at the cued location than when it is presented at an uncued location. Previous research has shown that IOR may be related to the oculomotor system. A number of studies have shown that saccades are inhibited from moving toward previously fixated locations (e.g., Klein & MacInnis, 1999; Abrams & Dobkin, 1994). Furthermore, Klein and Taylor (1994; Taylor & Klein, 1998; also see Rafal, Calabresi, Brennan & Sciolto, 1989) have proposed that IOR is tied to motor programming and that it does not necessarily require the actual execution of the response. According to Klein and Taylor, the programming of a saccade to a certain location results in inhibition of subsequent saccades to that location, irrespective of whether the saccade program results in the actual execution of the saccade.

In addition to examining the occurrence of IOR we also analyzed the saccade trajectories. If the execution of a saccade to the target requires a suppression of the onset-related activation, the trajectories of saccades to the target should reflect this. According to Tipper and colleagues, selection is achieved by inhibition of the neural population code activated by a distractor (e.g., Tipper, Howard & Paul, 2001). This distractor inhibition supposedly results in a sub-baseline level of activation at the distractor location. Since the displacement of the eyes is based on the mean vector of activity (e.g., Sparks et al., 1990) this inhibition should affect the saccade trajectory to the target; that is, the eyes should slightly curve away from the distractor location (e.g,. Tipper et al., 2001; Doyle & Walker, 2001; also see Sheliga et al., 1994; 1995).

The present study used a modified version of the oculomotor capture paradigm of Theeuwes et al. (1998, 1999). Observers had to make a goal-directed saccade to a uniquely colored target element while an irrelevant sudden onset was presented somewhere in the visual field. After fixating the initial target element, another element in the visual field became the next target element. This new target element was presented at the location at which the onset had previously appeared or at a location at which one of the other elements had appeared. If the onset activated an oculomotor program even when the eyes went directly to the target, it was expected that saccade latencies to the second target would be longer when it was presented at the onset location than when it was presented at one of the other locations. However, if the onset only activated an oculomotor program when the eyes initially moved in the direction of the onset, IOR was not expected when the eyes went directly to the target.

## Method

### Participants

Ten participants ranging in age between 18 and 28 years served as paid volunteers. All had self-reported normal or corrected-to-normal vision and reported having no color vision defects.

### Apparatus

A Pentium II Dell computer with a 21“ SVGA color monitor (Philips Brilliance 201 P) controlled the timing of the events and generated stimuli. Eye movements were recorded by means of an Eyelink tracker (SR Research Ltd.) with a 250 Hz temporal resolution and a 0.2° spatial resolution. The system uses an infrared video-based tracking technology to compute the pupil center and pupil size of both eyes. An infrared head motion tracking system tracked head motion. Even though head motion was measured, the head was stabilized by means of a chin rest. At the start of each trial when participants fixated the central fixation point, the eye position was automatically recalibrated to the center position to optimize the reliability of the eye movement measurements. After participants were well fixated on the central fixation point, they pressed a key to initiate a trial. Each participant was tested in a sound-attenuated, normally lit room, his or her head resting on a chinrest.

### Task

Participants viewed displays containing seven red-filled circles (1.3° in diameter) positioned on seven of eight equi-distant locations (one empty location) on an imaginary circle with a radius of 10° around a central fixation point (0.4°). Figure 1 gives an overview of the sequence of events. After 500 ms the fixation point turned from an asterisk into a plus shape (warning signal). After another 150 ms

an abrupt onset (a red circle having the same color as the other circles) was presented in the empty location. Fifty milliseconds later one of the circles turned gray, signaling the location to which a saccade had to be made (the first target). Another 800 ms later a second circle became gray constituting the next location to fixate. The first target remained present after presentation of the second target. Participants were instructed to first make an eye movement to the uniquely colored gray circle; they had to fixate this target until the second target was presented. Then they had to make a saccade toward the second target as soon as possible. Participants were told that the onset was irrelevant. They received no feedback about their performance. The colors of the circles, red and gray, were made equi-luminant (14.2 cd/m$^2$) and the circles appeared on a black background. The first target could be presented at one of four possible locations (either at 45°, 135°, 225°, or 315° angle). The sudden onset as well as the second target could be presented at either one of four other locations (either at 0°, 90°, 180°, or 270° angle).

On half of the trials the second target was presented at the location at which an onset was previously displayed or at a non-onset control location. Both locations were at the same distance and angle from the first target location (and the central fixation point). This guaranteed that differences in saccade latencies between these conditions could not be due to differences in the required saccade amplitude. On the other half of the trials the second target was presented at one of the other non-onset locations. There

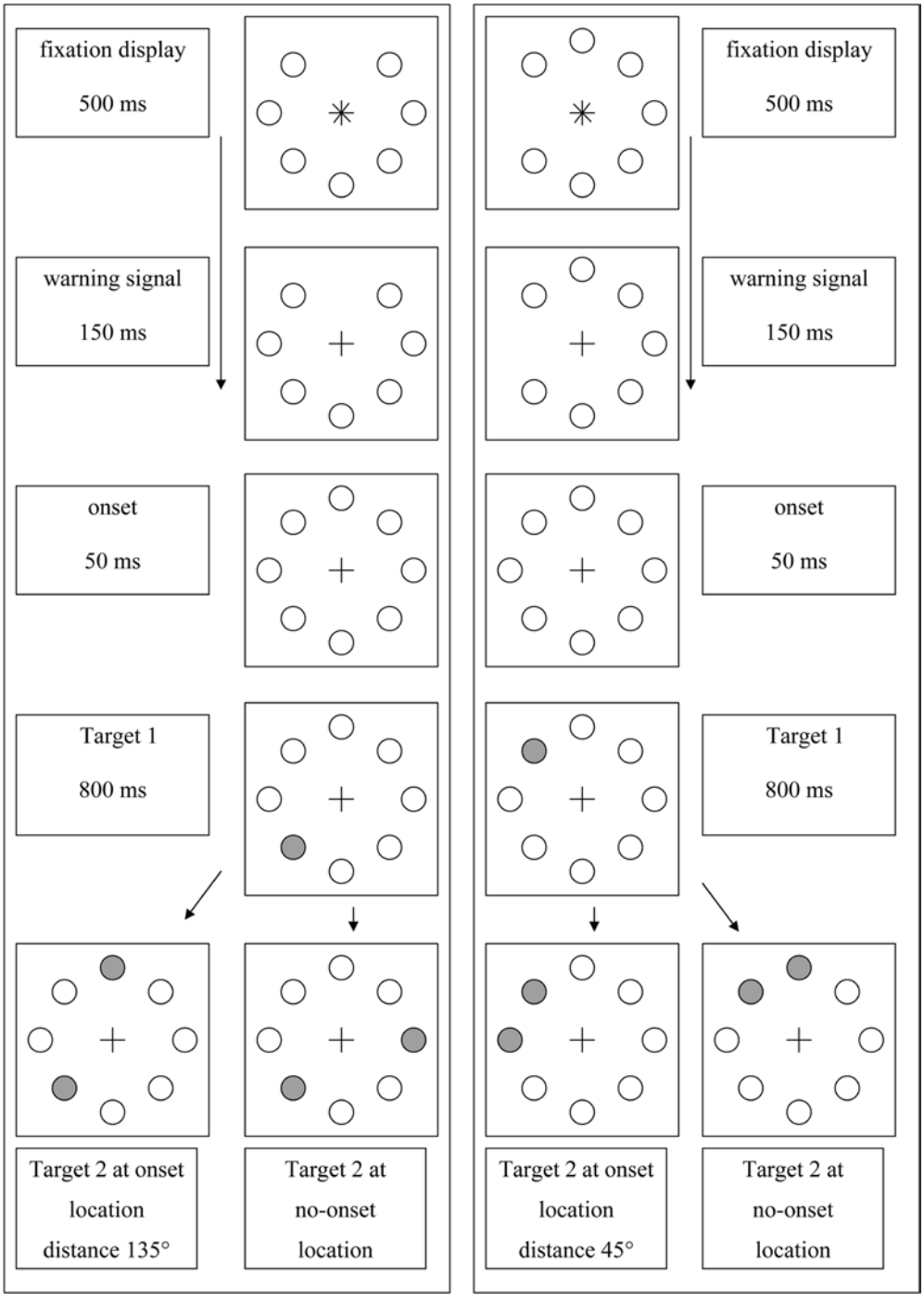


**Fig. 1** A graphic illustration of the displays and the temporal sequence of the experimental trials. The red circles are indicated by the *open circles*. The gray circles (the first and second target) are indicated by the *filled circles*

were 120 trials for each of the 8 combinations of angular distance between targets (45° or 135°) and the location of the second target (onset location, control location and two other locations) resulting in a total of 960 trials per participant. Figure 1 gives a graphic illustration of the displays.

## Results

Approximately 5.8% of the trials were discarded because observers moved their eyes too soon (within 80 ms of the presentation of the target). A further 6.6% of the trials were discarded because the first target was not fixated within a margin of 3°. Finally, 13.7% of the trials were discarded because the second target was not fixated within a margin of 3°. The initial saccade was assigned to a particular object if the endpoint of the initial saccade had an angular deviation of less than 22.5° (i.e., half the distance between neighboring objects) from the center of the object.

**Saccade endpoints** Figure 2 shows the distributions of the saccade endpoints of the initial saccades. As is clear from this figure, a large proportion of the initial saccades went towards the onset (36.2%). About 59.8% of the initial saccades went directly to first target.

**Saccade latency to the second target** The mean saccade latency to the second target (measured from the appearance of the second target) was calculated for trials on which the eyes first went to the onset (oculomotor capture trials) and for trials on which the eyes went directly to the first target (no oculomotor capture trials). Figure 3 shows the mean saccade latencies as a function of the location of the second target (second target at the onset location or at the equi-distant no-onset location) and angular distance between targets for 'oculomotor capture' trials and for 'no oculomotor capture' trials.

An analysis of variance (ANOVA) was performed on the saccade latencies for the second target with oculomotor capture (whether or not the eyes were captured by the onset), the location of the second target (onset or control) and the angular distance between targets (45° or 135°) as factors. Saccade latencies for the second target were longer when it was at the onset location (219 ms) relative to when it was at a control location (209 ms), $F(1,9) = 15.47$, $p < 0.005$, revealing IOR of about 10 ms at the onset location. No interaction was found between oculomotor capture and the location of the second target, $F(1,9) < 1$, indicating that IOR was not affected by whether or not the eyes initially went to the onset. Planned comparisons indeed showed a reliable IOR of 11 ms when the eyes moved to the onset ($p < 0.01$) and a reliable IOR of 10 ms when the eyes did not go to the onset ($p < 0.05$).

There was also a trend towards higher saccade latencies when the angular distance between targets was 135° (226 ms) than when it was 45° (203 ms), $F(1,9) = 4.04$, $p < 0.07$. No significant interactions were found between the location of the second target and the angular distance between targets, $F(1,9) < 1$, or between the location of the second target and oculomotor capture, $F(1,9) = 2.53$, $p > 0.14$; nor was there a

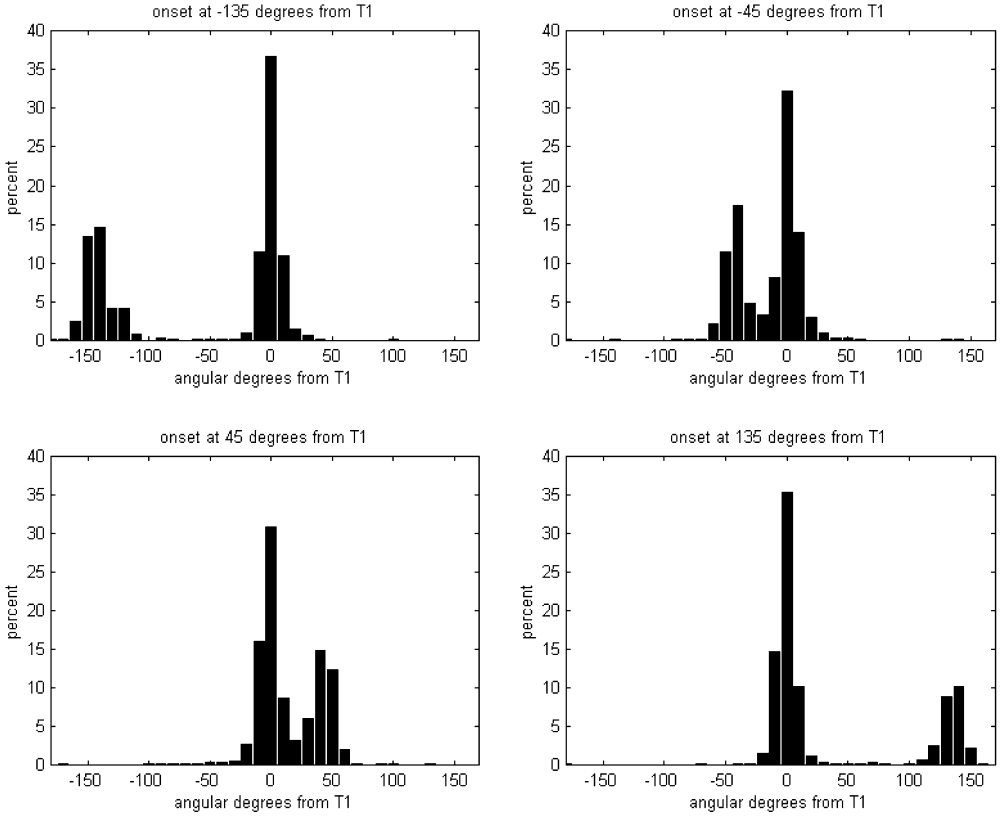


**Fig. 2** The distribution of the endpoints of the initial saccades in angular degrees from the first target (T1)

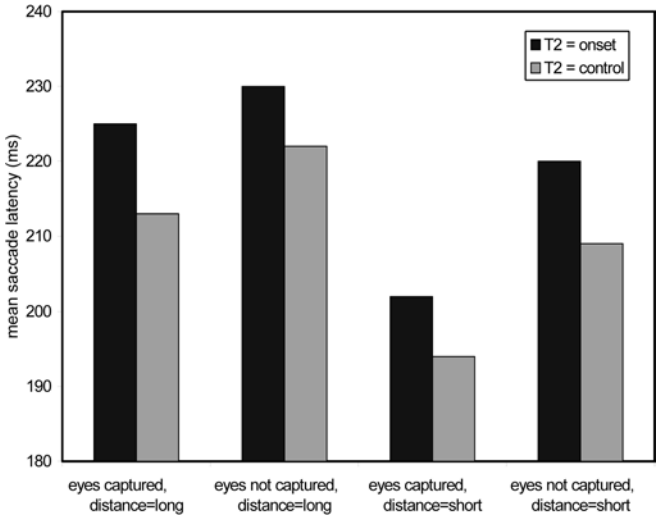


**Fig. 3** Mean saccade latency to the second target as a function of oculomotor capture (capture or no capture), the location of the second target (T2: onset or control) and the angular distance between targets (long = 135°, short = 45°)

three-way interaction between the location of the second target, the angular distance between targets and oculomotor capture, $F(1,9) < 1$. This indicates that the angular distance between targets did not significantly affect IOR.

**Saccade trajectories to the first target** In order to examine the trajectories of saccades to the first target we calculated for each 4 ms sample point the angular deviation of the saccade path relative to the required saccade path from fixation to the target. Figure 4 shows the distribution of the mean angular deviation of saccade trajectories to the first target. The saccade trajectories were divided into two parts (each containing half of the sample points) in order to illustrate the time course of the angular deviation of the trajectories. Figure 4a shows the distribution of the mean angular deviation of the first part of the saccade and Fig. 4b for the second part of the saccade. It is clear from Fig. 4a that onsets at –135° and 135° from the target produce quite similar distributions of mean angular deviation, at least in the central portion of the distributions. On the other hand, onsets at –45° and 45° result in distributions that are clearly shifted in the direction opposite the onset. In an additional analysis we determined whether this curvature in the direction opposite the onset was reliable. To this end we included only those saccades that initially went in the direction of the target (an angular deviation of less than 22.5° from the target at the second 4 ms sample after the eye was in motion). The analysis showed that the eyes were indeed shifted away from the onset: When the onset appeared at –45°, the mean angular deviation was 2.18°; when the onset appeared at 45°, the mean angular deviation was –2.00° ($t(1,9) = 4.17$, $p < 0.005$). This was not the case when the angular distance between target and onset was 135° ($t(9) < 1$).

Note in Fig. 4a, that at the extremes of the distributions a different picture emerges. A small proportion of saccades that ultimately end up at the target location start off as saccades towards the onset. Without any pause these saccades change direction in mid flight. These saccades have been labeled 'turnaround' saccades (Mokler & Fischer, 1999) or 'redirected' saccades (McPeek & Keller, 2001), since they appear to change their goal in mid-flight. Finally, Fig. 4b shows that the effect of onsets at –45° and 45° from the target on the distribution of the mean angular deviation is reduced for the second part of the saccade trajectory.

**Saccade trajectories to the onset** The trajectories of saccades to the onset were examined by calculating their mean angular deviation relative to the onset location. To examine the time course of the curvature the saccades were divided into two parts (each containing half the sample points) and the mean angular deviation relative to the onset location was examined for both parts of the saccades. See Table 1. An ANOVA was performed on the mean angular deviation relative to the onset location with side (the side of the target relative to the onset: negative/ anti-clockwise direction and positive/ clockwise direction), angular distance (45° and 135°) and section (first or second part of the saccade) as factors. There was a main effect of side, $F(1,9) = 25.76$, $p < 0.001$. The trajectories to the onset were curved in the direction of the target. There was also an interaction between angle and side, $F(1,9) = 11.05$, $p < 0.01$. The effect of side was greater at an angular distance of 45° than at an angular distance of 135°. Furthermore, there was an interaction between section and side $F(1,9) = 10.33$, $p < 0.01$. The effect of side was greater for the second half of the saccade than for the first half of the saccade.

**Saccade trajectories from the first to the second target** A final analysis of saccade trajectories was performed on the saccades from the first target to the second target. Since the onset could appear on both sides of the path between the targets when the distance between targets was long (135°) we only analyzed these trials. An ANOVA on the mean angular deviation of saccades to the second target with onset location as factor (–90°, 90°, 180° and 0°, i.e., onset at second target location) revealed a trend towards mean angular deviations away from the onset location, $F(3,27) = 2.10$, $p > 0.10$. In fact, a planned contrast between onsets on either side of the saccade path to the second target (–90° compared with 90° and 180° angular distance) was reliable, $t(9) = 2.66$, $p < 0.03$. This provides evidence that the onset location did have a small effect on the trajectory of the saccade from the first to the second target. At a 0° angular distance the mean angular deviation was –0.01°, at an angular distance of –90° this was 0.59° and at angular distances of 90° and 180° this was –0.76° and –0.85°, respectively.

In addition to these results concerning IOR and saccade trajectories, all additional findings were in line with Theeuwes et al. (1998, 1999). First of all, despite the fact that participants knew that the onset was

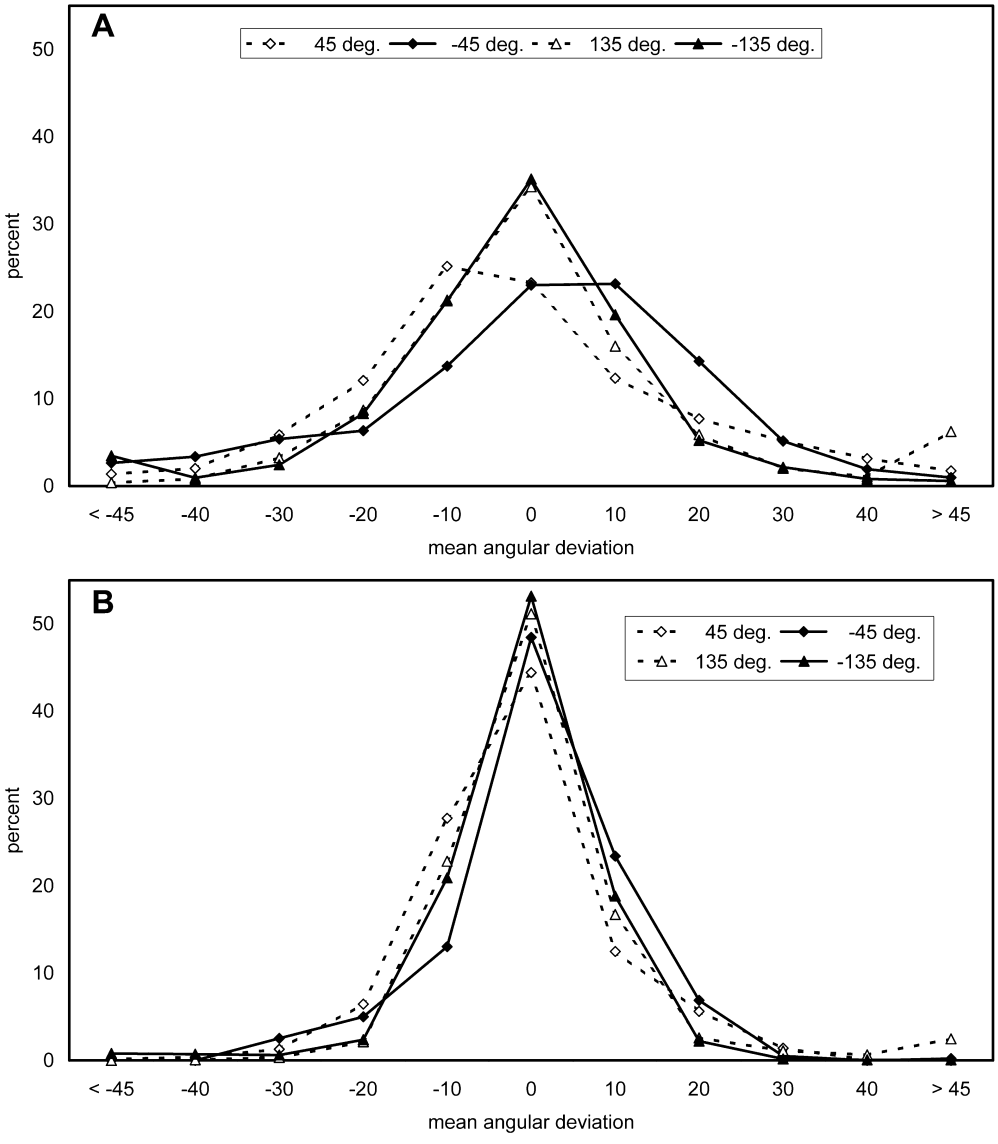


**Fig. 4A,B** The distribution of the mean angular deviation of saccades to the first target as a function of onset location. Saccades are divided into two parts (each containing half the samples) and the distributions are shown separately for the first (**A**) and second (**B**) part of saccades to the first target

**Table 1** Mean angular deviation of saccades to the onset as a function of saccade section (first or second part of the second) and target location.

| | Target location | | | |
|---|---|---|---|---|
| | –45° | 45° | –135° | 135° |
| first part of saccade | –2.48° | 0.29° | –1.45° | –1.14° |
| second part of saccade | –2.68° | 1.89° | –1.03° | 0.37° |

never the initial target, the eyes were captured by the onset on 36% of trials. Secondly, when the eyes were captured by the onset, they briefly stopped near the onset before moving on toward the first target (mean fixation duration 101 ms). Finally, latencies of initial saccades that went directly to the first target were longer (215 ms) than latencies of initial saccades that first went to the onset (157 ms), $t(9) = 9.70$, $p < 0.001$.

## Discussion

### Inhibition of Return

The goal of participants in our task was to make a goal-directed saccade towards the color singleton while a new, yet irrelevant object suddenly appeared elsewhere within the scene. On a large proportion of trials, the eyes did not directly go to the color singleton target; instead the eyes were captured by the onset, that is, the eyes first moved to the onset before they moved to the color singleton target. On these oculomotor capture trials, subsequent orienting to the location of the sudden onset was slowed: it took longer to make a saccade to an object presented at the location that previously contained the onset, than to another location in the visual field. This finding is in line with previous findings and shows that the eyes are inhibited from returning to a location that has already been fixated (e.g., Abrams & Dobkin, 1994; Kingstone &

Pratt, 1999; Klein & MacInnis, 1999; Taylor & Klein, 1998). Importantly, however, even when the eyes were not captured by the sudden onset, but went directly to the color singleton target, there was also IOR of saccades to the location of the onset. This suggests that even when the eyes did not first move to the onset, inhibition of the location at which the onset appeared must have occurred. In other words, regardless of whether or not an actual reflexive saccade to the onset was made, subsequent orienting to this location was inhibited.

### Saccade trajectories

Further evidence that onsets affected the oculomotor system even on trials on which the eyes went directly to the target was provided by analyses of the saccade trajectories. When the angular distance between target and onset was 45° the trajectories of saccades to the target were often slightly curved in the direction opposite the onset. When the angular distance was 135° no curvature away from the onset was found. If we assume that the curvature in the opposite direction of the onset is the result of an inhibition of the onset-related activation (e.g., Tipper et al., 2001) then this effect of angular distance reflects a greater change in the mean vector of activity by inhibition of locations close to the target relative to inhibition of locations far from the target (e.g., Doyle & Walker, 2001).

In addition to the effect of onset location on the saccade trajectory to the first target, some preliminary evidence was found that the onset location also affected the subsequent saccade trajectory from the first to the second target. To our knowledge this is the first evidence that the effect of onsets on saccade trajectories is not restricted to the first saccade after presentation of the onset, but can also be found in subsequent saccade trajectories. Admittedly the effect of the side of the onset relative to the path towards the second target was small, but this is not surprising given the relatively large angular distance between onset and second target location (90° and 180°).

Finally, analyses of saccade trajectories toward the onset revealed an effect of target location. These saccades often curved towards the target. In fact, on a small proportion of trials the eyes started to move toward the onset, but turned around in mid-flight and ended near the target location. These 'turnaround' or 'redirected' saccades have been reported in previous studies (e.g., Mokler & Fischer, 1999; Schlag-Rey et al., 1997). These results suggest that after the onset-related activation reaches the threshold required for saccade execution, top-down target-related activation starts to rise in the saccade programming map resulting in a deviation of the saccade path in the direction of the target.

## Saccade Programming

In order to understand how the onset affects the oculomotor system it is important to examine how saccade programming is accomplished. A wide range of studies have identified the fundamental role of the superior colliculus (SC) in the programming of saccades (for reviews see Schall, 1991; Wurtz et al., 2000; Sparks & Mays, 1981). The SC is a structure in the midbrain, which receives input from a wide variety of areas such as the frontal eye fields (FEF), the posterior parietal cortex (PPC), the striate cortex (V1) and directly from the retina. In turn it projects to the brainstem premotor circuitry to trigger saccadic eye movements (Moschovakis, 1996). It has been suggested that the intermediate layers of the SC integrate exogenous and endogenous input for saccade programming (Trappenberg et al., 2001). The intermediate layers of the SC contain at least three types of cells: fixation cells, build-up cells and burst cells (Munoz & Wurtz, 1993). When a saccade is programmed, the build-up neurons start to show low frequency activity (Dorris et al., 1997) and according to Trappenberg et al. (2001) burst neurons receive a strong inhibition until the activity in the build-up neurons reaches a certain threshold. Both build-up and burst neurons have a high frequency burst of activity when saccades are executed. Fixation neurons, on the other hand, are active during fixation and are passive when saccades are executed. Krauzlis et al. (1997) have shown that fixation cells can basically be considered as build-up neurons with a foveal receptive field. Together, build-up and fixation cells form a saccade map in which eye movements are based on the vector average of the activity in the build-up and fixation cells (e.g., Wurtz et al., 2000). An important feature of this saccade map is that there is substantial evidence for short distance excitation and long distance inhibition within the SC (e.g., Olivier et al., 1998; Munoz & Istvan, 1998). Within the SC saccade programming can basically be considered as a competition between activity at different locations of the saccade programming map (e.g., Trappenberg et al., 2001). Thus, activity related to different objects in the visual field is mutually inhibitory.

### Exogenous and endogenous saccade control

In accordance with Trappenberg et al., (2001) we assume that exogenous and endogenous activation is integrated in a saccade programming map in the intermediate layers of the SC. However, the control signals related to these two modes are to some extent different. On the one hand, exogenous activation reaches the intermediate layers of the SC through a network involving the lateral intraparietal area (LIP) in the PPC and areas in the visual cortex as well as the superficial layers of the SC which receives direct input from the retina and projects to LIP (e.g., LaBerge, 1995). On the other hand, endogenous activation reaches the intermediate layers of the SC through a cortical network including the LIP and visual cortex and also a number of areas in the prefrontal cortex such as the supplementary eye fields (SEF), the dorso-lateral prefrontal cortex (DLPFC) and

most notably the FEF (Schall, 1991). Together with the other oculomotor areas, the SC, FEF and LIP form a network responsible for saccade programming. Although the present discussion will focus on the role of these three oculomotor areas, it must not be forgotten that other areas (such as the SEF and DLPFC) also play their own specific role.

The role of the FEF in endogenous saccade control has been well-documented (e.g., Schall, 1991; Schlag-Rey et al., 1992). The FEF has excitatory connections to the SC through which the FEF can provide the SC with a spatial code of the saccade goal. Furthermore, the FEF is capable of inhibiting location-specific activation within the SC through inhibitory links through the caudate and substantia nigra (e.g., Passingham, 1993). The inhibitory role of the FEF is primarily based on lesion studies, which have shown that FEF lesions result in a deficit in inhibiting reflexive saccades (Guitton, Buchtel & Douglas, 1985; Henik, Rafal & Rhodes, 1994; Rafal et al., 2000). Furthermore, electrical micro-stimulation of the FEF results in suppression of saccades in a variety of tasks (Burman & Bruce, 1997). The inhibitory control signals from the FEF to the SC could play an important role in saccade programming. Since saccades are based on the mean vector of activity (e.g., Sparks et al., 1990) inhibition of irrelevant locations could prevent the execution of inaccurate saccades.

LIP is part of the PPC, which contains a number of areas which hold modality-specific spatial representations of movement goals (e.g., Andersen et al., 2000). Apart from LIP, which is involved in saccade control, the other main motor control areas in PPC are the parietal reach region (PRR), involved in reaching and the anterior parietal region (AIP), involved in grasping (Sakata et al., 1997). It has been claimed that the PPC holds short-term representations of spatial goals and that these representations are manipulated by areas in the prefrontal cortex (e.g., FEF and DLPFC) which supposedly perform modality-specific executive operations (e.g., Rao et al., 1997; also see Schneider, 1999).

## Oculomotor suppression hypothesis of IOR

As discussed in the previous section the location-specific inhibition from the FEF to the SC may play a mayor part in saccade programming. Inhibiting irrelevant locations facilitates the programming of a saccade to a target object and avoids the execution of an inaccurate saccade. It has been proposed by Tipper and colleagues (e.g., Tipper et al., 2001; Howard & Tipper, 1997; also see Doyle & Walker, 2001) that inhibition of distractor-related activation results in a sub-baseline level of activation at the distractor location. Furthermore, since saccade displacement is based on the mean vector of activity this inhibition results in the execution of a saccade which curves away from the distractor location. This is consistent with the results of the present study. In addition to the effects on saccade trajectories we propose that the inhibition of irrelevant locations outlasts the initial saccade and results in delayed saccades to the location of the distractor (IOR).

Our account of IOR, which we call the oculomotor suppression hypothesis of IOR therefore assumes that IOR is the result of a location-specific inhibition to bias oculomotor programming to a goal location. The inhibition occurs both to move away from fixation as well as to move the eyes to one location and not another. That is, in order to execute a saccade to a specific goal location activity at fixation needs to be inhibited as well as activity at other irrelevant locations. According to the present account IOR and saccade trajectory curvature away from a distractor location arise from the same source, namely location-specific oculomotor suppression. It must be noted that at present this hypothesis is rather speculative and further research is needed to test it. For example, we predict that the curvature of saccades away from irrelevant distractors persists for some time (as does IOR), at least beyond the execution of the first saccade. We found some preliminary evidence for this, but this clearly needs further testing. Also, we predict that after moving to a specific location subsequent saccades should be curved away from this previously fixated location.

## Neural correlates of IOR

According to the oculomotor suppression hypothesis, neural correlates of IOR should be found in SC and possibly FEF. Since the SC and FEF are part of an oculomotor network including LIP, it is possible that the location-specific inhibition of irrelevant locations also spreads to LIP either from SC or from FEF. This would facilitate the coordination between these areas and avoid conflicting signals being produced. There is clear evidence supporting the role of the SC in IOR. The first line of evidence comes from patients with progressive supranuclear palsy (PSP), a neuro-degenerative disorder of the SC and surrounding areas. In PSP patients generation of voluntary saccades was impaired and they showed a loss of IOR along the axis where their saccade deficits were most severe (Posner et al., 1985; Rafal et al., 1988). Also, single cell recording studies of monkeys engaged in a cue-saccade paradigm showed that targets presented at cued locations elicited reduced neural activity relative to targets presented at uncued locations (Dorris et al., 1999). It is possible that this reduced neural activity was the result of inhibition from other areas such as the FEF or LIP. As suggested above it might have been the inhibitory connections from FEF through the substantia nigra to SC which caused the reduced activity. In fact the substantia nigra is also severely affected in PSP, so it is possible that the degeneration of the substantia nigra contributes to the loss of IOR in these patients. Another single cell recording study suggests that IOR may also be reflected in the FEF, although this was not recognized by the authors.

Hanes, Patterson and Schall (1998) required monkeys to fixate a central fixation point and to execute a saccade to a peripheral target which appeared simultaneously with the offset of the fixation point. On some trials, after a variable delay period, the central fixation point re-appeared, signaling the monkey to withhold the saccade. Hanes et al. found reduced activity in 50% of the FEF cells with visually evoked activity when a saccade was successfully withheld relative to activity of these cells on latency-matched no-stop-signal trials. Importantly, this activity reduction came well after the time needed to inhibit the saccade and therefore we tentatively suggest that this activity reduction might reflect IOR in these FEF cells. Given the suggestion that IOR is the result of inhibition from the FEF on neuronal activity in the SC it is not necessary to assume that IOR should be found in the FEF itself. However, it is possible that IOR is fed back to the FEF either from the SC or from LIP, or alternatively it may be the result of inhibitory interneurons within FEF.

There is also some evidence that IOR may also be found in LIP. In a single cell recording study in LIP Robinson, Bowman, and Kertzman (1995) found reduced cell activity when a saccade target was presented at a cued location relative to when it was presented at an uncued location.

These studies provide some initial support for the view that IOR is spread throughout a network of oculomotor centres, including SC, FEF and LIP. IOR in SC seems well-established, but more neurophysiological evidence needs to be gathered to determine whether IOR can also be found in areas such as FEF and LIP.

## Oculomotor suppression in the present study

The results of the present study fit nicely with the oculomotor suppression hypothesis.

Prior to the presentation of target and onset subjects fixate the central fixation point. Activity in the saccade programming map is highest at fixation (see Fig. 5a). After the presentation of the static color singleton target together with the onset, activity first increases at the onset location, due to the faster time course of exogenous input in the SC (e.g., Trappenberg et al., 2001). At this point fixation activity is reduced due to the lateral inhibition from the onset-related activity (see Fig. 5b). Subsequently, target-related input arrives in the SC activating the neurons which have the target in their receptive field. Target-related activity, onset-related activity and fixation-related activity are then all mutually inhibitory. Since participants have the goal of executing a saccade to the color singleton target and must ignore the onset, the FEF may inhibit onset-related activity as well as fixation-related activity in order to execute an accurate saccade to the target (see Fig. 5c). In trials in which the eyes move directly to the color singleton target the inhibition of the onset location results in a curvature of the saccade away from the onset and delays subsequent saccades to the location of the onset (IOR). However, when the initial activation of the onset location is sufficient to reach threshold before the inhibition from the FEF reaches the SC, a saccade will be directed to the onset. In these trials the onset location is inhibited in basically the same way as in trials in which the eyes move directly to the target. The only difference is that the inhibition sets in too late to prevent the saccade to the onset. Thus, after the eyes move on from the onset to the target subsequent saccades to the onset location are also delayed due to the inhibition of the onset location.

## Other support for the oculomotor suppression hypothesis

The oculomotor suppression hypothesis of IOR makes a number of other predictions. As mentioned above, the suppression of the onset location sometimes occurs too late to stop the eyes from moving in the direction of the onset. However, it may be that some target-related activity has already arrived in the SC saccade programming map at the moment the threshold required for saccade execution has been reached. Since the saccade destination is based on the average vector of activity, the target-related activity will result in a significant undershoot of the saccade to the onset. Obviously this will only occur if target-related activity in the SC occurs before the saccade to the onset is completed. Indeed, we found that saccades to the onset were slightly curved in

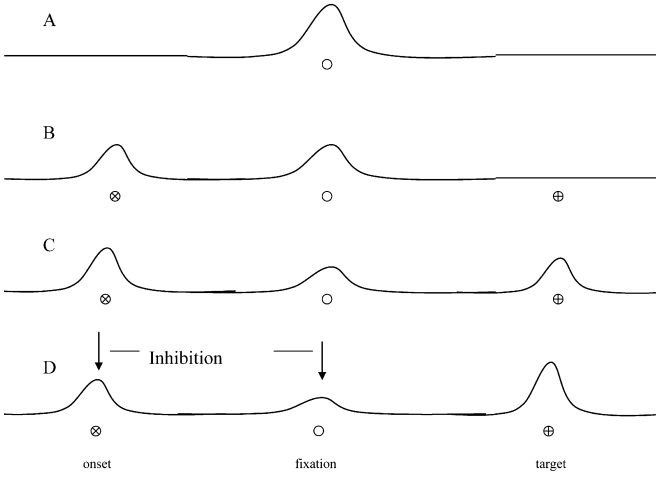


**Fig. 5** Activity in the saccade map illustrating the competition between target, onset and the fixation location. (*A*) Prior to the presentation of target and onset subjects actively fixate the fixation point. There is a strong fixation-related activity. (*B*) Soon after presentation of target and onset, activation related to the onset arrives in the saccade map. Due to lateral inhibition from the onset, activity at fixation is reduced. (*C*) Somewhat later target-related activation arrives in the saccade map. Activation at the target location, onset location and fixation location is all mutually inhibitory. (*D*) In addition to the lateral inhibition, top-down inhibition is applied to the onset and fixation locations, biasing saccade programming towards the target location

the direction of the target and this curvature was greatest in the latter part of the saccade. Furthermore, the continuing inhibition of the onset location together with the activation of the target location could result in extremely short fixation durations on the onset. Consistent with these predictions, a number of studies using the oculomotor capture paradigm have shown that saccades to the onset often stop well before reaching the onset and mean fixation durations were typically around 100 ms as in the present study (e.g., Theeuwes et al., 1998, 1999; Godijn & Theeuwes, 2002). In the present study, as well as in other studies using similar tasks, it has even been found that the eyes occasionally start moving to an onset and in mid-flight turn around and move toward the goal location (e.g., Mokler & Fischer, 1999; Shlag-Rey, Amador, Sanchez & Schlag, 1997). Since target-related activation increases as a function of time it may also be expected that the amplitude of the saccade to the onset and the subsequent fixation duration are inversely related to the latency of the initial saccade to the onset. Specifically, the higher the latency of saccades to the onset, the higher the target-related activity and the shorter the amplitude and fixation duration on the onset should be. This is precisely what we recently found (Godijn & Theeuwes, 2002). Thus, sometimes the inhibition of the onset location prevents the execution of the saccade to that location, otherwise it results in a short fixation duration and possibly a significant undershoot. In both cases, after the saccade to the target, subsequent saccades to the location of the onset are delayed.

The oculomotor suppression hypothesis of IOR can also explain results of typical cue-target IOR studies (e.g., Posner & Cohen, 1984). When a peripheral cue is presented this activates an oculomotor program to that location. Since the cue has to be ignored and subjects are required to remain fixated in the center, inhibition is applied to the location of the cue. It takes a certain amount of time before activation related to the location of the cue can be inhibited. Thus, activation starts to increase shortly after cue onset and eventually decreases due to the inhibition. If the cue-target SOA is short, programming a saccade to the target should benefit from the activation of the onset. However, if the cue-target SOA is long, the inhibition should result in a delayed saccade. Since the PPC is also involved in other motor systems, the oculomotor suppression in the SC, flowing through the PPC, could also inhibit other responses. In fact, Tipper et al. (2001) have shown that the curvature away from a to-be-ignored stimulus is affected by whether subjects are required to reach towards the target. According to Tipper et al., this cross-talk between specific motor modalities may occur in the PPC. In the same manner, IOR may also be passed between modalities. Thus, the typical finding that manual responses are inhibited when a target is presented at the cued location at long SOAs (e.g., Posner & Cohen, 1984) could also be an effect of oculomotor suppression. An alternative possibility is that IOR may have several causes. IOR may originate in other motor systems apart from the oculomotor system and in some conditions it may also originate in the attentional system. Future research should test the generality of the oculomotor suppression hypothesis.

## Oculomotor suppression and attention

Our account of IOR is similar to that of Klein (e.g., 1988, 2000; Klein & Taylor, 1994; Taylor & Klein, 1998) in the sense that IOR is generated in the oculomotor system. However, initial accounts of IOR suggested that IOR is due to a discouraging of the re-orienting of attention to a previously cued location (Posner & Cohen, 1984). According to this line of reasoning, the abrupt onset captures attention exogenously and this will cause IOR at the location of the onset regardless of whether an eye movement is actually executed. Such an interpretation is in line with earlier findings that show that abrupt onsets have the ability to capture attention exogenously (e.g., Theeuwes, 1991, 1994; Yantis & Jonides, 1984). One problem with the attentional account is that endogenous shifts of attention do not result in subsequent IOR (e.g., Posner & Cohen, 1984). Thus, one must assume that only exogenous shifts of attention result in IOR. Since events that elicit exogenous shifts of attention also tend to initiate oculomotor programming it is hard to claim that IOR is the result of exogenous shifts of attention. On the other hand, programming an endogenous saccade does result in IOR (whether or not the saccade is actually executed; e.g., Rafal et al., 1989). However, there are a number of reasons to believe that IOR is also somehow related to attention. First, IOR can be object-based. That is, if a peripheral cue is moved to a different location, IOR is found at the location to which the cue has moved (e.g., Tipper et al., 1991; Abrams & Dobkin, 1994). Second, IOR is also found in non-spatial discrimination tasks (e.g., Lupianez et al., 1997; Pratt et al., 1997). These findings suggest that IOR may also have an effect on the attentional system. One possible explanation for these findings is that in addition to oculomotor IOR there is also IOR in the (exogenous) attentional system (e.g., Kingstone and Pratt, 1999). However, given the presumed role of the posterior parietal cortex (PPC) in IOR it is possible that the attentional effects merely reflect the relation between PPC and the perceptual system. The PPC is a major source of input for areas in the temporal and visual cortex, in particular IT and V4. These areas are part of what is typically known as the ventral stream in which featural information is processed. Area V4 is specialized in processing of object features, while IT is specialized in processing complex objects (Tanaka, 1993). According to LaBerge (1995) (also see Hahn & Kramer, 1998) activation from PPC flows through the pulvinar of the thalamus to V4 and on to IT and enhances activation at the selected regions while suppressing activation at surrounding regions. In other words, attentional selection

of relevant locations is mediated by PPC. Thus, if the PPC is indeed involved in IOR it is possible that IOR in the PPC is passed on to structures involved in object recognition, which would explain the attentional effects of IOR.

A strong relationship between the attentional system and the motor system is consistent with previous research showing that performance on letter discrimination tasks is best when the discrimination target is presented at the saccade target (e.g., Deubel & Schneider, 1996; Kowler, Anderson, Dosher & Blaser, 1995; Hoffman & Subramaniam, 1995). In fact, it has been argued that the attentional system provides the oculomotor system the spatial control signals for exogenous and endogenous saccades (Schneider & Deubel, *in press*). According to Schneider's VAM model (1995) there is a unitary selection mechanism with two goals: selection for perception on the one hand and selection for action on the other. Selection-for-action is achieved through the dorsal stream from V1 to the PPC (e.g., Schneider, 1995; Schneider & Deubel, *in press*). In parallel with this, selection-for-perception is achieved in the ventral stream from V1 to the inferior-temporal cortex (IT). According to VAM, motor programming is a consequence of visual attention. An alternative account of the relationship between attention and eye movements is Rizzolatti's premotor theory of attention (e.g., Rizzolatti et al., 1987). According to Rizzolatti et al., attention shifts are made on the basis of eye movement programs. The oculomotor suppression hypothesis seems more compatible with the premotor theory; IOR originates in the oculomotor system and from LIP it affects perception in the ventral stream (IT and V4) through its descending pathways, in the manner described by LaBerge (1995). However, it is also possible that attentional IOR is not the result of oculomotor suppression, but instead may originate in the attentional system itself. In that case it is still possible that the control signals flow from the attentional system to the motor system instead of vice versa.

## Summary

In summary, the present study showed that when subjects were searching for a color singleton target a sudden onset captured the eyes in a large proportion of trials and resulted in IOR of subsequent saccades to the location of the onset. Whether or not a saccade to the onset was executed prior to the saccade to the target had no effect on the size of the inhibition. We suggest that IOR is the result of the inhibition of saccade-related activity necessary to move away from the onset or to avoid executing a saccade in its direction. We further suggest that a network of structures is involved in IOR, specifically the FEF, the SC and the PPC. IOR may also affect the perceptual system through the interaction between the motor systems and the perceptual systems, mediated by the selection of relevant locations in the PPC. It must be noted that these suggestions are still rather speculative and that further research must be awaited to determine more precisely the relationship between effects of IOR on the oculomotor system and its effects on the perceptual system. Furthermore, more neurophysiological evidence needs to be gathered to further support the hypothesized neural correlates of IOR.

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